Review

Time To Inject Some Contrast into the Nephrotoxicity Debate

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Iodinated contrast media have long been feared for causing contrast-induced AKI (CI-AKI), a concern rooted in early reports with high-osmolar agents. Experimental data suggest potential nephrotoxic mechanisms, yet clinical evidence from older uncontrolled studies was confounded by comorbidities and procedural risks. Contemporary propensity-matched and controlled analyses consistently show that modern low-osmolar and isoosmolar contrast agents (mostly intravenously administered) uncommonly cause true nephrotoxicity, even among high-risk populations, such as patients with advanced CKD, AKI, or critical illness. Large randomized trials, including the Prevention of Serious Adverse Events Following Angiography trial, found no difference in outcomes between sodium bicarbonate and isotonic saline hydration as preventive strategies, nor *N*-acetylcysteine administration, and highlighted risks like fluid overload. Persistent fear of CI-AKI has fueled renalism: unnecessary avoidance or delay of essential imaging, leading to worse outcomes. Current consensus emphasizes individualized care—avoiding hypovolemia, limiting contrast dose, and withholding nephrotoxins only in severe kidney impairment. A balanced, evidence-based approach should replace outdated caution. For decades, clinicians have approached iodinated contrast media with excessive caution over CI-AKI. This concern originated in the mid-20th century when a seminal 1954 case report described acute renal failure in a patient with myeloma after injection of a high-osmolar contrast agent. Early contrast formulations indeed had nephrotoxic effects *via* intense renal vasoconstriction, osmotic diuresis, and direct tubular injury. By the 1980s, any AKI after contrast exposure was broadly labeled CI-AKI, typically defined by a postprocedure increase in serum creatinine (>0.5 mg/dl or >25%) in the absence of other causes. Transition to safer low-osmolar and isoosmolar agents in the late 1980s appears to have been a pivotal step, with other factors likely contributing to the temporal decline in CI-AKI incidence. These include refinement of AKI definitions, improvements in preventive measures (avoiding hypovolemia and suspension of nephrotoxic medications), reduction in the amount of injected contrast, more cautious patient selection, and overall improvements in the management of the underlying conditions for which contrast is administered (*e.g.*, acute coronary syndromes and surgical and perioperative care). Nevertheless, the historical fear of CI-AKI has persisted, often disproportionate to modern evidence, sometimes leading to avoidance of necessary diagnostic imaging. This perspective reexamines the nephrotoxicity debate in light of current knowledge, aiming to replace outdated dogma with a balanced, evidence-based approach that prioritizes patient outcomes over unfounded fears.

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Evidence of Contrast Nephrotoxicity: Mechanisms and Observational Data

Laboratory and animal studies provide biologic plausibility for contrast-induced kidney injury.[1] *In vitro*, iodinated contrast can directly damage renal tubular cells and vascular endothelium, causing cellular swelling, vacuolization, and apoptosis. Unlike contrast media, water is reabsorbed in the renal tubules, resulting in increased intratubular concentration of contrast, along with greater osmotic load and viscosity. Elevated viscosity increases vascular resistance and slows medullary blood flow. In addition, contrast agents dysregulate the synthesis and release of vasoactive mediators—raising endothelin and adenosine levels while reducing nitric oxide and prostaglandin

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expression—thereby promoting vasoconstriction. Together with the redistribution of blood flow from the medulla to the cortex during ischemia, these mechanisms exacerbate medullary hypoxia.[2] These effects can trigger oxidative stress, mitochondrial dysfunction, and tubular cell necrosis.[3] Not surprisingly, animal models given high doses of iodinated contrast show acute tubular injury with epithelial cell shedding and intraluminal obstruction. Such findings support a potential for nephrotoxicity, especially with older high-osmolar agents—indeed, high-osmolality contrast was far more cytotoxic to kidney cells than modern low-osmolality formulations.[4] However, osmolality alone is unlikely to account for this difference because multiple studies have failed to demonstrate a significant variation in contrast-induced AKI (CI-AKI) incidence between isoosmolar and low-osmolar formulations.[5] It should be kept in mind that the route of contrast administration may also have an effect: intra-arterial injection with first-pass renal exposure (*e.g.*, from the left heart, thoracic or suprarenal abdominal aorta, or the renal arteries) results in a higher contrast concentration than either intravenous administration or intra-arterial injection with second-pass renal exposure. Clinical observations have also linked contrast administration with AKI in specific contexts, particularly when high volumes of contrast agents were used.[6,7] However, it should be emphasized that higher contrast dose is often correlated with more complex and prolonged procedures, longer intra-arterial manipulation, and overall greater patient fragility. These factors may themselves increase the risk of AKI and are rarely accounted for in dose-response studies, potentially inflating the apparent nephrotoxic contribution of contrast. Many uncontrolled studies reported postcontrast creatinine rises in 10%–30% of patients, especially among those with preexisting CKD or diabetes. Early landmark studies in the cardiac catheterization laboratory found a roughly 14% incidence of AKI after coronary intervention and noted that this CI-AKI was associated with worse survival. Such observational data cemented the notion that iodinated contrast was a leading cause of hospital-acquired AKI, even being cited as a top culprit in some series.[2] In clinical practice, it became common to blame contrast whenever kidney function declined after imaging, an assumption reinforced by these associative findings.

However, critical appraisal reveals major limitations in the evidence implicating contrast. Most older studies lacked proper control groups, in which everyone received contrast, so any postprocedure AKI was attributed to it by default. Patients referred for contrast-enhanced studies often have more comorbidities or hemodynamic instability (*e.g.* acute coronary syndromes) than those who avoid contrast, introducing selection bias. In addition, the current definition of CI-AKI (a small creatinine rise within 48 hours) is inherently nonspecific because such changes can result from numerous factors including dehydration, hemodynamic instability, or concomitant nephrotoxic exposures—an important limitation common to all AKI settings. In retrospective studies, much of the reported CI-AKI may have simply been contrast-associated AKI (coincidental kidney injury around the time of imaging) rather than CI-AKI.[8] This distinction is crucial—correlation does not prove causation. Indeed, with controlled comparisons, the relative risk from contrast often disappears. For example, a meta-analysis of intraveinous (IV) contrast studies that included patients with CKD found no significant difference in AKI rates between those who received contrast and well-matched patients who did not.[9] The authors concluded that these retrospective studies failed to show renal damage attributable to contrast in patients with CKD, although they cautioned that residual confounding could still be present. In summary, although susceptibility mechanisms for contrast nephrotoxicity are real, the clinical magnitude of risk in modern practice appears far smaller than once feared, particularly with the more recent contrast formulations.

Modern Clinical Studies Challenge the CI-AKI Dogma

Over the past decade, a growing body of clinical data have challenged the traditional CI-AKI paradigm. Large propensity score–matched analyses in diverse settings have consistently found that iodinated contrast exposure does not independently heighten AKI risk in most patients. For instance, McDonald *et al.* studied over 53,000 patients undergoing computed tomography (CT) scans and detected no difference in post-CT AKI incidence between those who received IV contrast and those who had unenhanced scans, including patients with advanced CKD, even if they represent only a small part of the cohort.[10] Absence of contrast nephrotoxicity has also been reported in a large retrospective database study,[11] although this analysis presents several important limitations, including reliance on International Classification of Disease coding from discharge charts, potential unmeasured confounders, and the inclusion of a broad population not specifically enriched for patients at higher risk of contrast associated-AKI. In this study,[11] a surprising statistical protective association was observed with contrast exposure, which most likely reflects methodologic artifacts, including selection bias, unmeasured confounders, and collider bias, rather than a true protective effect. Lee *et al.* performed a meta-analysis explicitly focused on patients with CKD and found no significant contrast-attributable risk of AKI in that high-risk group.[9] In short, multiple reviews agree that patients with normal renal function have essentially zero risk of CI-AKI, and even those with moderate CKD (eGFR down to approximately 30 ml/min) have minimal risk.[8] It is important to emphasize that, for an equivalent decline in GFR, the resulting increase in serum creatinine appears much greater in patients with preexisting CKD than in those with preserved kidney function, due to the inverse and nonlinear relationship between GFR and serum creatinine, which becomes particularly steep at lower GFR levels. Any measurable nephrotoxic effect of modern low-osmolality contrast seems largely confined, if it exists at all, to the most severely reduced GFR range.[8]

Reassuring evidence extends even to populations traditionally considered to be at an extremely high risk of CI-AKI. Two recent studies illustrate this point. Ehmann *et al.* examined over 14,000 hospitalized patients who already had AKI on presentation, comparing those who received contrast-enhanced scans with those who did not. After propensity adjustment, contrast administration had no

association with worsened kidney outcomes—AKI recovery by discharge and 6-month dialysis rates were identical whether patients received contrast.[12] Likewise, Berglund *et al.* studied critically ill intensive care unit patients undergoing CT and found no increase in AKI incidence or long-term renal decline in those who got iodinated contrast, compared with those who underwent noncontrast CT.[13] The adjusted odds ratio for AKI with contrast showed no risk increase in that fragile intensive care unit cohort. These findings are striking—even among patients with tenuous kidney function or critical illness, iodinated contrast did not meaningfully worsen renal outcomes on average. However, it should be acknowledged that propensity-matched studies are not equivalent to prospective randomized controlled trials (RCTs); in the specific context of contrast nephrotoxicity, several such studies have suggested residual selection bias and often lacked adjustment for preexisting AKI or the use of prophylactic strategies before contrast exposure.

Nevertheless, some paradoxical patterns in previous literature now appear more understandable. For example, the risk of CI-AKI was long thought to jump markedly once eGFR drops below certain thresholds (*e.g.* <60 or <30 ml/min), and intra-arterial contrast (such as during angiography) was deemed more nephrotoxic than IV contrast for CT. One of the plausible reasons could be the direct vasoconstrictive effect of contrast on the renal vasculature.[14] That aside, iodinated contrast is not metabolized by the liver and is excreted almost entirely unchanged by the kidneys regardless of the administration route. Therefore, the contrast agent's intrinsic effect on renal tissue should be similar whether given IV or intra-arterially. The likely explanation for reported differences is that patient factors and cointerventions drive much of the AKI risk. For instance, cardiac catheterization involves arterial manipulation that can shower atherosclerotic plaque debris into the renal circulation which itself causes AKI independent of any contrast chemical toxicity.[15] This complication is unique to intra-arterial procedures and could falsely amplify the perceived nephrotoxic risk of contrast in those settings. Likewise, an abrupt rise in risk at a certain eGFR cutoff probably reflects the fact that patients with very low eGFR are sicker overall (and possibly managed more cautiously, creating referral bias), rather than a sudden change in contrast's biologic effects. In short, many noncontrast factors—hypotension, ischemia, microembolism, and concurrent nephrotoxins—conspire to cause AKI during complex procedures and severe illness. Modern evidence suggests that once these confounders are accounted for, the isolated contribution of iodinated contrast to AKI is minimal in most cases. Crucially, traditional CI-AKI risk factors (*e.g.*, advanced age, diabetes, existing CKD, heart failure, nephrotoxic drugs, and hypotension) align closely with general hospital-acquired AKI risk factors.[16,17]

Strategies To Prevent CI-AKI for All Patients: Time To Recognize Futility!

Driven by the significant concern regarding CI-AKI at the time, numerous preventive strategies became the focus of extensive and valuable research. More than 40,000 patients were enrolled in various RCTs that failed to demonstrate clear clinical benefit for interventions such as intravenous isotonic crystalloid administration, *N*-acetylcysteine, and statins. The Prevention of Serious Adverse Events Following Angiography (PRESERVE) trial,[18] using a 2×2 factorial randomized clinical trial design, demonstrated no difference in outcomes between sodium bicarbonate and isotonic saline, nor between *N*-acetylcysteine and placebo, among patients considered to be at a high risk of nephrotoxicity. Among randomized trials, A Maastricht Contrast-Induced Nephropathy Guidelines Study (AMACING)[19] uniquely compared prophylactic intravenous hydration with no hydration in guideline-targeted high-risk outpatients (eGFR 30–59 ml/min per 1.73 m^2) undergoing elective procedures across both intravenous and intra-arterial routes, finding noninferiority of no hydration with fewer complications and lower costs. By contrast, Prevention of Contrast Renal Injury With Different Hydration Strategies (POSEIDON)[20] compared two hydration strategies after an identical preprocedure bolus in a single-center cardiac catheterization setting using intra-arterial contrast and left ventricular end diastolic pressure-guided titration; although the lLVEDP-guided arm showed lower creatinine-based CI-AKI rates than fixed-rate hydration, these findings do not establish the necessity of hydration versus no hydration and may reflect hemodilution and setting-specific factors. Nevertheless, it should be emphasized that patients with an eGFR below 30 ml/min per 1.73 m^2 were excluded from the AMACING trial[19] and that the effectiveness of such a strategy in this particularly sensitive population remains to be addressed.

Clinical Implications and Path Forward

The evolving evidence base has prompted a reevaluation of prophylactic strategies and clinical decision making around contrast use. A 2020 joint consensus statement by the American College of Radiology and National Kidney Foundation emphasized that the AKI risk from iodinated contrast has been overstated and should not automatically preclude necessary imaging.[8] The consensus advises that for patients with severely reduced kidney function (eGFR <30 or those on acute dialysis), preprocedural isotonic crystalloid administration, or, more importantly, to avoid hypovolemia, is reasonable to mitigate any potential risk. However, it stresses that for the vast majority of patients, especially those with mild-to-moderate CKD, the risk of true contrast-induced nephrotoxicity is very low, and decisions should focus on the clinical necessity of imaging. In practical terms, this means that we should avoid reflexively canceling or delaying contrast-enhanced scans out of fear and instead perform an individualized risk-benefit analysis. Delaying diagnosis (or opting for inferior imaging modalities) in a patient with treatable pathology poses its own serious hazards.[21] Continued belief in CI-AKI profoundly influences clinical decisions, resulting in diagnostic delays, unnecessary and potentially harmful isotonic crystalloid administration protocols, and selection of suboptimal imaging alternatives.[21] The phenomenon known as renalism significantly contributes to increased morbidity and mortality. Chertow *et al.* highlighted the detrimental consequences of reduced

coronary angiography referrals among patients with CKD experiencing myocardial infarction, driven by exaggerated nephrotoxicity fears and resulting in increased mortality.[21] As Berglund *et al.* note, clinicians must weigh the substantial diagnostic/treatment benefits of indicated contrast studies against the now-demonstrated lack of significant nephrotoxic harm in most scenarios.[13] In many cases, the scale tips decisively in favor of getting the study and managing any manageable risks, rather than abstaining from contrast.

Of course, prudent precautions remain appropriate for high-risk individuals. Keeping patients well volume-expanded, minimizing contrast dose, and avoiding overlapping nephrotoxics are sensible measures to further reduce the already-low odds of renal injury. Technologic innovations are also enabling necessary imaging with dramatically less contrast. For example, in coronary interventions for patients with advanced CKD, operators can leverage intravascular ultrasound and other adjuncts to perform ultra-low-contrast or even zero-contrast angioplasty, as demonstrated in the Minimizing cOntrast utiliZAtion With IVUS Guidance in coRonary angioplasTy (MOZART) trial (which achieved a median contrast volume of only approximately 20 ml with an image-guided technique).[22] Interestingly, in this study, the incidence of CI-AKI was not significantly increased.

Such approaches are important for rare patients whose kidneys truly cannot tolerate standard contrast loads. In general, however, the evidence indicates that fear-driven avoidance of contrast has been far more detrimental than contrast itself. Unwarranted deferral of CT scans or angiography may lead to missed or delayed diagnoses, suboptimal therapy, and worse patient outcomes—the very scenario we hoped to prevent.

Conclusion

It is time for the medical community to inject some evidence-based realism into the contrast nephrotoxicity debate. Iodinated contrast media, particularly modern low-osmolality agents, are considerably safer for the kidneys than traditional teaching suggests. Contrast is not entirely benign, and vigilance is warranted particularly in patients with severely impaired renal function (eGFR <30 ml/min per 1.73 m^2). In this population, it is advisable to prevent hypovolemia, to avoid or temporarily interrupt concomitant nephrotoxic medications when feasible, and to minimize both the volume of contrast administered and the number of procedures requiring contrast. If a patient develops AKI, a close longitudinal follow-up of renal function is warranted to detect potential long-term complications.

Conversely, an overly cautious approach that treats all contrast media as inherently harmful may lead to delays in diagnosis and suboptimal management. Therefore, a careful evaluation of the individual risk-benefit ratio remains essential. By acknowledging the limitations of past studies and embracing contemporary data (including consensus guidelines), we can recalibrate our approach. The goal moving forward should be to ensure that patients who need contrast-enhanced imaging—even those with CKD—receive it without inappropriate delay, accompanied by reasonable precautions rather than prohibitive fear. Such a balanced, data-driven strategy may help reduce unnecessary hospital stays and costs, decrease the likelihood of diagnostic errors, and ultimately contribute to improved patient outcomes. In the end, the greatest risk may come not from the contrast itself but from not using contrast when it is clinically indicated.

Disclosures

Disclosure forms, as provided by each author, are available with the online version of the article at http://links.lww.com/KN9/B327.

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